APPENDIX H

NATIONAL POLLUTION DISCHARGE ELIMINATION SYSTEM HUMAN HEALTH RISK ASSESSMENT

Introduction

This Appendix presents a human health risk assessment in support of Proposed Action to direct Y-12 West End Treatment Facility (WETF) treated effluent to the Oak Ridge City sewer system. This alternative is discussed in Section 2.2.1 of the Environmental Assessment. Currently, the Y-12 WETF liquid wastes are treated in a Five-step process to

- 1 Remove heavy metals and radionuclides,
- 2 Remove nitrates,
- 3 Remove organic compounds, and
- 4 Remove solid particulates
- 5 Make final adjustments to the liquid at Effluent Polishing System (EPS).

After the Five step process is completed effluents are sampled and released into Upper East Fork Poplar Creek (UEFPC) through a permitted National Pollution Discharge Elimination System (NPDES) outfall. Due to improvements in the WETF system (i.e., addition of step 1), the need for EPS has been significantly reduced. The proposed action described in section 2.1.1 therefore, includes releasing treated WETF effluent into the Y-12 and City of Oak Ridge sewer systems after the Four step treatment process. Treated waters will be analyzed for 165 Priority Pollutants (40 CFR 136) to verify compositions meet proposed sewer release limits (See Environmental Assessment *Appendix B, Table B.12*). Those batches not meeting sewer release limits or found to be otherwise unsuitable for release to the sewer will be sent to the EPS for further treatment and released to the NPDES outfall at UEFPC.

The purpose of this Appendix is to model the human health risk impact of changing the ultimate disposition of WETF effluents. Currently, treated effluent is released at the NPDES outfall into UEFPC. Under the proposed action, WETF treated effluent will be released to the Y-12 Sewer System, undergo further treatment with other municipal sewage and be released at the City's NPDES outfall at Lower East Fork Poplar Creek (LEFPC). This assessment conservatively models the relative risk to human health by 1) releasing treated WETF effluents at the NPDES outfall at UEFPC and 2) releasing the WETF effluents to the Oak Ridge city sewer system and releasing the treated effluents at the city's permitted outfall.

The risk assessment evaluates a hypothetical child exposed to creek water of UEFPC and LEFPC through wading. The approach and methodology used in this human health risk assessment are consistent with the guidance developed by the National Research Council (NRC). The NRC, established by the National Academy of Sciences to further scientific knowledge and to advise the federal government, developed the four-step paradigm for conducting health-based risk assessments (NRC 1983). This paradigm has been adopted by EPA as well as many other federal and state agencies. In accordance with the NRC recommendations, this risk assessment is organized into the following four steps: 1) Identification of Constituents of Concern (COCs), 2) Toxicity Assessment, 3) Exposure Assessment and 4) Risk Characterization

Identification of COCs

The COCs modeled in this study are listed in *Table H.1*. These comprise the metals, organic compounds, inorganic compounds, and radionuclides specified in the WETF NPDES permit and in the proposed list of constituents to be released from WETF into the city sewer system.

Table H.1. Concentrations of constituents used in risk assessment

	able 11.1. Concentration	s of constituents used in risk a	issessinent
	WETF NPDES	Maximum Detected	Predicted
Constituents	Outfall	Concentration at	Concentration at City
	Concentration	WETF Outfall to	of Oak Ridge NPDES
	Limits to UEFPC	UEFPC (mg/liter)	Outfall
	(mg/liter)		(mg/liter)
METALS			
Arsenic	0.052	0.026	0.00002
Cadmium	0.15	0.2	0.00001
Chromium	1.0	0.03	0.00008
Copper	1.0	0.03	0.00022
Lead	0.20	0.8	0.00008
Mercury	0.20	0.1	0.00004
Nickel	3.98	2.85	0.00016
Silver	0.50	0.03	0.00008
Zinc	2.0	0.6	0.00056
ORGANIC COM	IPOUNDS		
Benzene	0.01	0.01	0.00002
Methylene	0.01	0.01	0.00004
Chloride			
Phenols	0.01	0.01	0.00048
Toluene	0.01	0.01	0.00002
TCE	0.01	0.01	0.00003
INORGANIC CO	OMPOUNDS		
Cyanide	1.2	0.03	0.00007
RADIONUCLID	DES		
Total	0.096	0.048	0.0035
Uranium			

Toxicity Assessment

The toxicity assessment identifies the relationship between the magnitude of exposure or dose and the potential for occurrence of specific health effects or responses for each COC. Both carcinogenic and noncarcinogenic effects are considered. Dose response values for chemicals are derived from the Integrated Risk Information System (IRIS). IRIS is an EPA maintained webbased electronic data base, containing the most recently updated information on human health effects resulting from exposure to various chemicals. Dose response values for uranium are taken from *Health Risks From Low-level Environmental Exposure to Radionuclides, Federal Guidance Report No. 13 Part I – Interim Version* (EPA 1998).

Non carcinogenic effects are evaluated using the EPA accepted Reference Dose (RfD) for ingestion and inhalation of specific chemicals. EPA has develop both chronic and subchronic RfDs. A chronic RfD is defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound. Chronic RfDs are used to evaluate the potential non carcinogenic effects associated with exposure periods between 7 years (approximately 10 percent of a human lifetime) and a lifetime. As noted in the next section this assessment assumes an exposure duration of 9 years and, therefore, utilizes chronic RfDs.

Table H.2. summarizes the dose-response information for the COCs with potential non carcinogenic effects for the oral and inhalation routes of exposure reported in the IRIS data base. For each chemical, the dose-response value, and the reference for the dose-response value is presented. In addition, the target organ and critical effect upon which the dose-response value is based are also presented for each chemical.

The underlying assumption of a risk assessment for constituents with known or assumed potential carcinogenic effects is that no threshold dose exists; consequently, there is an underlying assumption that a finite level of risk is associated with any dose greater than zero. The EPA methodology is to extrapolate dose-response relationships observed at the relatively high doses used in animal studies to the low dose levels encountered by humans in environmental situations.

The mathematical models assume no threshold and use both animal and human data to develop a potency estimate for a given compound. The potency estimate, called a cancer slope factor (CSF), is expressed in units of (mg/kg-day)⁻¹ for chemical carcinogens. *Table H.3.* summarizes the oral and inhalation dose-response information reported in IRIS for potentially carcinogenic COCs identified for this assessment.

The EPA considers all radioactive elements to be cause both cancer and genetic mutation. The risk, however, of serious genetic effects is much lower than the risk of cancer (EPA 1989); therefore, this assessment considers the carcinogenic effects of radioactive constituents only. EPA developed slope factors for radionuclides are expressed as (pCi)⁻¹ for the ingestion and inhalation routes and in various forms for external exposure to ionizing radiation, including m³/pCi-second for immersion, m²/pCi-second for ground plan exposure, and kg/pCi-second for exposure to soils of a given activity of radioactive constituent.

Table H.2. Dose-response data for COCs with potential noncarcinogenic effects

CAS ^a	
Secondary METALS	
METALS	
METALS Arsenic 7440382 NAb 3.0E-4° Liver, Kidney, Skin Cadmium 7440439 NA 5.0E-4 Resp. System, Kidney Chromium-VI 7440473 2.29 5.0E-3 Skin Chromium-III 7440473 NA 1.5E+0 Skin Copper 7440508 NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidneys <th></th>	
Arsenic 7440382 NAb 3.0E-4° Liver, Kidney, Skin Cadmium 7440439 NA 5.0E-4 Resp. System, Kidney Chromium-VI 7440473 2.29 5.0E-3 Skin Chromium-III 7440473 NA 1.5E+0 Skin Copper 7440508 NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108883 1.14 2.0E-01 CNS, Liver, Kidneys	
Cadmium 7440439 NA 5.0E-4 Resp. System, Kidney. Chromium-VI 7440473 2.29 5.0E-3 Skin Chromium-III 7440473 NA 1.5E+0 Skin Copper 7440508 NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS ^e , CNS Chloride Phenol 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01 E-01 CNS, Liver, Kidneys	
Chromium-VI 7440473 2.29 5.0E-3 Skin Chromium-III 7440473 NA 1.5E+0 Skin Copper 7440508 NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS ^c , CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidneys E-01 CNS, Liver, Kidneys E-01	
E-6	s, Prostate, blood
Chromium-III 7440473 NA 1.5E+0 Skin Copper 7440508 NA NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS ^e , CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidneys and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys	
Copper 7440508 NA NA Gastrointestinal Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS ^e , CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys	
Lead 7439921 NA NA CNS ^d ; blood Mercury 7439976 8.57 NA Respiratory System, K E-5 E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys	
Mercury 7439976 8.57 NA Respiratory System, K Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys	
E-5 Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	
Nickel 7440020 NA 2.0E-2 Lungs, CNS, Paranasa Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	idneys, CNS,
Silver 7440224 NA 5.0E-3 Nasal Septum, Skin, E Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	
Zinc 7440666 NA 3.0E-1 Blood; anemia ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	l Sinus
ORGANIC COMPOUNDS Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	yes
Benzene 71432 NA NA Blood, CNS, Skin, Bo Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	
Methylene 75092 NA 6.0E-2 Skin, CVS°, CNS Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01 E-01 E-01 E-01	
Chloride Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	ne, Marrow
Phenol 108952 NA 6.0E-01 Liver, Kidney and Ski Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	
Toluene 108883 1.14 2.0E-01 CNS, Liver, Kidneys E-01	
E-01	n
Triallana de la la Titolo NA NA Desciore Corto I	
Trichloroethylene 79005 NA NA Respiratory System, h	eart, liver, CNS
INORGANIC COMPOUNDS	

^a Chemical Abstracts Service Registry Number.

^b NA = Not available; inhalation RfD is not listed in EPA IRIS database 2/01.

 $^{^{}c}$ RfDs are from EPA IRIS database 2/01

 $[^]d$ CNS = Central Nervous System.

^eCVS = Cardiovascular System

Table H.3. Dose-response data for COCs with potential carcinogenic effects

Compound	CAS^a	Weight of evidence ^b	Oral slope factor (mg/kg-day) ⁻¹	Inhalation slope facto (mg/kg-day) ⁻¹
METALS				
Arsenic	7440382	A	1.5E+0	1.51E+1
Cadmium	7440439	B1	NA^c	6.3E+0
Chromium-VI	7440473	A	NA	4.2E+1
Copper	7440508	D	NA	NA
Lead	7439921	B2	NA	NA
Mercury	7439976	D	NA	NA
Nickel	7440020	A	NA	NA
Silver	7440224	D	NA	NA
Zinc	7440666	D	NA	NA
ORGANIC COMPO	OUNDS			NA
Benzene	71432	A	5.5E-2	NA
Methylene Chloride	75092	B2	7.5E-3	NA
Phenol	108952	D	NA	NA
Toluene	108883	D	NA	NA
Trichloroethyle ne	79005	NA	NA	NA
INORGANIC COM	IPOUNDS			
Cyanide	57125	D	NA	NA

^aChemical Abstracts Service Registry Number.

^bWeight of Evidence Classifications:

A=Human carcinogen (sufficient evidence of carcinogenicity in humans)

B1=Probable human carcinogen (limited evidence of carcinogenicity in humans)

B2=Probable human carcinogen (sufficient evidence of carcinogenicity in animals, with inadequate or lack of evidence of carcinogenicity in humans)

C=Possible human carcinogen (limited evidence of carcinogenicity in animals, and inadequate or lack of evidence of human data)

D=Not classifiable as to human carcinogenicity

^cNA = Not available

Table H.4. Dose-response data for Uranium carcinogenic effects

Compound	Weight of evidence	Oral Slope Factor (pCi) ⁻¹	External Exposure Slope Factor L(pCi-yr) ⁻¹
Uranium-235-D	A	4.7E-11	4.1E-16
Uranium-238-D	Α	6.2E-11	8.3E-19

Exposure Assessment

Exposure is defined as the contact of a human with a chemical or physical agent (EPA 1988a). The magnitude of exposure is determined by measuring or estimating the amount of an agent available at the exchange boundaries (i.e., the lungs, gut, skin) during a specified time period. The exposure assessment is the determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, and route of exposure.

The purpose of developing this exposure model is to assess the change in potential risk to human health associated with releasing WETF effluents at the City's discharge point on LEFPC as opposed to releasing them at the Y-12 discharge point on UEFPC.

The hypothetical receptor considered for exposure to the WETF effluents is a child wading in the UEFPC and LEFPC below the WETF and the City's respective NPDES discharge points. Because access to the Y-12 site is restricted it is unlikely that a child could be exposed to waters on the reservation; however, much of the creek is outside the reservation boundaries. The concentration of constituents in the creek at offsite locations will be rapidly and significantly diminished through dilution as they migrate downstream from the WETF discharge point. In this assessment, however, it is conservatively assumed that there is no dilution at offsite locations (i.e., we are assuming exposure at the release point at Y-12). Risk is therefore estimated for the a child exposed to water containing concentrations defined for the WETF NPDES outfall limits (*Table H.1.*).

Similarly, estimated risks to a hypothetical child wading in LEFPC are based on the modeled outfall concentrations at the discharge point with no dilution from stream water. It is conservatively assumed that all (100%) the mass (metals, uranium, etc.) from WETF sewer discharge point is released to the City's outfall after being joined by Y-12's other sewer inputs and the city of Oak Ridge's input.

The chemical intake model is documented in *Table H.5.* All assumptions are based on EPA recommended values or highly conservative assumptions (e.g., 3 hour wading events, 36 event/year, 9 years of exposure). The dominant exposure routes are assumed to be 1) incidental ingestion of water containing metal, organic compounds, inorganic compounds, and uranium, 2) inhalation of volatile organic compounds, and 3) exposure to ionizing radiation from uranium. It is assumed that this is no reasonable inhalation exposure route for metals, including uranium, in the wading scenario since all metals other than mercury have vanishingly small vapor pressures. The vapor pressure of mercury is also orders of magnitude less than that for benzene (~10⁻³ torr) and at the dilute concentrations considered in this model (1-0.03 mg/liter) its partial pressure will approach zero.

Table H.5. Intake Models for a trespassing child wading in Upper East Fork Poplar Creek

Parameter (unit)	Values	Reference	
Contact rate (milliliters/hour)	50	EPA (1988) Superfund Exposure Assessment Handbook	
Inhalation rate (meter ³ /hour)	1.9	EPA (1997) Exposure Factors Handbook. Rate for children involved in "heavy" activity.	
Exposure Time (hours/event)	3	Conservative judgement	
Exposure Frequency (events/year)	36	Conservative judgement based on a wading event occurring 3 days/week over the a 12 week period. The national average for swimming is 7 days/year (EPA 1988)	
Exposure Duration (years)	9	National median time at one residence (EPA 1989) Exposure Factors Handbook	
Body Weight (kilograms)	24.6	EPA (1997) Exposure Factors Handbook. This is a conservative minimum weight. Assuming 9 years of exposure from age 7 to 16 the range in body weight is 24.6 kg for a girl age 7 to 66.8 kg for a male age 16	
Noncarcinogen Averaging Time (days)	3285	Exposure duration in days	
Carcinogen Averaging Time (days)	25550	EPA (1989) Risk Assessment Guidance for Superfund: Volume 1,	
Volatilization Factor (liters/ meter³)	0.5	EPA (1991) Risk Assessment Guidance for Superfund: Volume I, P	

Equations for ingestion and inhalation of chemicals in water, respectively are:

$$Intake (mg/kg-day) = \frac{CW \times CR \times ET \times EF \times ED}{BW \times AT} \qquad Intake (mg/kg-day) = \frac{CW \times K \times IR \times ET \times EF \times ED}{BW \times AT}$$

Equation for ingestion of uranium in water:

$$Intake (pCi) = AW \times CR \times ET \times EF \times ED \times CF$$

where: CW = chemical concentration in water (milligram/liter),

AW = activity of uranium in water (pCi/liter),

CR = contact rate (liters/hour),

IR = inhalation rate (cubic meters/hour)

K = volatilization factor (liters/cubic meter)

ET = exposure time (hours/event),

EF = exposure frequency (events/year),

ED = exposure duration (year),

BW = body weight (kilogram), and

AT = averaging time (day)

Risk Characterization.

In the risk characterization step, the results of the exposure assessment are combined with the results of the toxicity assessment to derive pathway-specific quantitative estimates of potential health risks. The estimates for each exposure pathway are then summed to give total risk estimates. Separate quantitative estimates of potential risk are derived for potentially carcinogenic effects and for noncarcinogenic effects.

The potential health effects for non carcinogens is modeled by the Hazard Quotient (HQ). The HQ is ratio of the modeled intake of the COC to the RfD. Intakes that exceed the RfD, or an HQ greater than one indicates the potential for an adverse human health. The combined potential health effects of the COCs is estimated by the Hazard Index (HI), the simple sum of HQs for all COCs. An HI greater than one is defined as the level of concern for potential adverse noncarcinogenic health effects (EPA 1989).

Cancer risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of pathway-specific exposure to carcinogenic COCs. Results of the cancer risk estimates can be compared with the acceptable risk range of 10⁻⁶ to 10⁻⁴ that is the goal of EPA outlined in the National Oil and Hazardous Substances Pollution Contingency Plan (40 CFR 300).

The risk to an individual resulting from exposure to chemical or radiological carcinogens is expressed as the increased probability of a cancer occurring over the course of a lifetime. The increased cancer risk is calculated by estimating the daily intake of a chemical carcinogen averaged over a lifetime multiplied by a contaminant-specific CSF. Oral and inhalation pathway-specific CSFs have been derived for certain carcinogens; some carcinogens do not have a CSF available or are presently under review by EPA. All CSFs used in the chemical risk estimate calculations were obtained from IRIS.

The CSF converts estimated daily intakes averaged over a lifetime of exposure directly to the incremental risk of an individual developing cancer (EPA 1989). The carcinogenic risk estimate is generally an upper-bound estimate because the CSF is typically derived as the upper 95% confidence level of the probability of response based on experimental animal data (EPA 1989). Thus, EPA is reasonably confident that the "true risk" will not exceed the risk estimate derived through use of the CSF and is likely to be less than that predicted using CSFs (EPA 1989).

Table H.6. summarizes the modeled health effects of a child wading in UPEFC at the WETF Outfall as compared to the same child wading at the city's LEFPC outfall. The risk to the a child wading at either outfall is less the EPA target range of 10⁻⁴ to 10⁻⁶ for acceptable risks levels.

The estimated carcinogenic risk at the WETF outfall is 8.2 x 10⁻⁷, if it is assumed the NPDES outfall releases at it's permitted limits and is 5.6 x 10⁻⁷ if the risk is modeled on the maximum concentration measured at the outfall. The risk estimated if the mass of COCs is released to the sewer system and all COCs are released through the city's NPDES outfall is 5.1 x 10⁻⁹. The latter estimate assumes that all mass released into the sewer system is at the proposed WETF sewer discharge limits. This latter value is two orders of magnitude less than the value modeled for the WETF outfall.

The hazard index for exposure to COCs are summarized in *Table H.6.* The HI for both ingestion and inhalation pathways is less than the EPA threshold of one at both outfalls. The HI at the WETF outfall, assuming all releases are at the permit limit for all COCs, is between 0.71 to 0.51. This range is based on the valence state of chromium, the former value estimated assuming all is in the hexavalent state. The HI at the WETF outfall, calculated assuming all releases are at the maximum measured concentrations of all COCs, is 0.17. The valence state chromium has less of an impact at maximum measured outfall concentrations because its concentration is two orders of magnitude below the discharge limit. The HI calculated assuming all mass released at the WETF sewer discharge point is at the proposed sewer discharge limits and all mass is released at the LEFPC outfall is 0.0001. This value is four orders of magnitude below the EPA threshold of 1 and three orders of magnitude less that the HI modeled for the WETF outfall.

Table H.6. Modeled Health Effects

Health Effects	UEFPC Y-1	2 OUTFALL	LEFPC CITY OUTFALL	
	NPDES- Limits	Maximum Release	Modeled on Sewer Release Limits	
Hazard Index (All Cr-IV)	0.71	0.17	0.00014	
Hazard Index (All Cr-III)	0.51	0.17	0.00012	
Risk (Non Radiological)	7.4E-07	5.0E-07	7.9E-10	
Risk (Uranium)	1.0E-07	5.0E-08	4.1E-09	
Total Risk	8.4E-07	5.5E-07	4.9E-09	

References

EPA 1988, Superfund Exposure Assessment Handbook, Office of Emergency and Remedial Response. EPA/540/1-88/001

EPA 1989, Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A). EPA/540/1-89/002.

EPA 1990, *Exposure Factors Handbook*. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-89/043.

EPA 1991, Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors, Office of Solid Waste and Emergency Response, OSWER Directive 9285.6-03, Washington, D.C., March 25.

EPA 1992, *Dermal Exposure Assessment: Principles and Applications*. EPA, Office of Research and Development. EPA 600/8-91/011A.

EPA 1994 *Integrated Risk Information System (IRIS)*, Health and Environmental Criteria and Assessment Office, Cincinnati, Ohio.

EPA 1995, Health Effects Assessment Summary Tables, Table 4: Radionuclide Carcingenicity - Slope Factors, Office of Emergency and Remedial Response, Washington D.C.

EPA 1997, Exposure Assessment Handbook, Office of Emergency and Remedial Response, Washington D.C.